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A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Dynamics and Management of Para Partum Neuropathic Reactions

VICTOR M. VICTOROFF, M.D.
Cleveland, Ohio

History of Study

The first report which Dr. Joseph L. Fetterman and I prepared on the subject of psychosis associated with childbirth was presented at the meeting of the American Psychiatric Association in May, 1949.[1] At that time we presented data from a study of eighty-six patients who developed psychotic illness in relationship to pregnancy. We introduced the term "para partum psychosis" to designate such mental illness as appeared in the literature under the heading "gestational," "post-partum," "puerperal," and "lactation" psychosis.

We noted at that time two main groups of patients:

1) Those who suffered an acute illness, developing abruptly during earlier days after parturition, running a stormy course with a tendency toward favorable outcome.

2) A more insidious type which began either during pregnancy or at any time up to six months after delivery with features hardly distinguishable from schizophrenia.

We cited Zilboorg's[2] statement echoed by Deutsch that hostility towards the baby is a nodal point in the symptom complex of post partum psychosis but emphasized the self-destructive impulses which constitute a major danger in the handling of these patients. There were 34 in that earlier series (25.3%) referred for psychiatric help only after a serious threat or attempt at suicide had been made. Two patients neglected by their families killed themselves and one who made recovery during treatment became a suicide at a later date (2.8%).

The prompt use of electrocoma therapy was appreciated as being surprisingly effective, particularly in the acute reaction types, showing excellent recovery in over 90 percent of this group irrespective of the diagnostic category in which they had been placed. This experience of course is quite different from what is usually attained with shock therapy in which there is a spectrum of effectiveness, being most useful in depressions and least in schizophrenics from whom affect has been leached.

A second paper was prepared in October, 1949.[4] By that time 100 case histories had been collected. Para partum psychosis was defined as a clinical entity, "With well established onset, chartable course, and variable outcome," and we stated, ". . . it is characterized by alternative symptoms as readily distinguished as any in neuropsychiatric classification."

The patients were described as falling into certain symptom categories represented as, "depressed," "phobic," and "schizophrenic." The further comment was made that, "Other patterns appeared where the major motif was hypochondriasis, obsessive compulsive ritualism, psychopathic behavior, manic disorders or combinations of these."

Presented at the meeting of the Cleveland Society of Neurology and Psychiatry, Wednesday, November 21, 1951.
From the Fetterman Clinic, Cleveland, Ohio.

By this time the prevailing sense of inadequacy, anxiety, self-doubt and guilt which appeared almost universally among the patients was evaluated. In a tentative discussion of etiology it was underlined that: *"At the central core of the para partum psychosis regardless of the numerous causative peripheral factors is the urge to annihilate the child,"* again verifying the psychoanalytic concept suggested by Zilboorg.

We touched on the possibility of organic determinants and listed cerebral anoxia, fluctuations in endocrine physiology, subclinical toxemia, exhaustion states and changes in water balance. At that time information was too scanty to adjudicate between victims of neuropathic determinants from those purely organic or environmental in origin.

It was already clear that whatever primary causes there may have been for para partum psychosis, in the presence of a constellation of secondary factors the illness should be feared. These were listed as: personality predisposition, physical predisposition and present stress. Addressed as it was to the obstetricians the paper included an admonition to the audience to pay more attention to mothers with the "maternity blues" and gave some recommendations for the psychic support of the pregnant woman and manipulation of her environment to reduce the danger of physical and emotional stress patterns.

Origin of Data

This essay is buttressed by experience with 190 cases of this syndrome who have suffered 225 separate instances of puerperal psychosis among them. In order to utilize this material as effectively as possible 55 patients who have been personally treated and followed have been selected for the discussion. This group has been subjected to intense psychotherapy, many for a period of two to three years. There is little doubt that these 55 patients are representative of the entire group, since their assignment to me was in each instance purely random and fortuitous.

In the survey of material each patient is described according to the presenting syndrome which prevailed at the time of her acute illness. (Chart I.) Then separately listed are the manifest symptoms and signs, (Chart II) and physical complications (Chart III).

Predeterminate factors are divided into hereditary-familial (Chart IV) and developmental categories, which latter includes neuropathic traits, and early psychic trauma (Chart V). A section of situational effectors immediate to the pregnancy is also catalogued (Chart VI).

The protocol contains over 200 individual entries, any one of which has been suspected singly or in combination to be the cause of para partum psychosis and emerges in the series here presented. The importance in the individual case of each factor has been weighed and the frequency with which each factor appears has been separately listed.

As yet no central group of factors emerges as *sine qua non* for the provocation of the para partum psychoses. Living through a complete parturition experience culminating in the birth of a viable child is not required; women in our series have suffered varieties of the symptom-complex after abortions and miscarriage. It is even dubious if implantation and conception are necessary in view of the fact that three patients in our larger series developed full blown psychoses after adopting children. Perhaps more remotely we may beg the question of whether this disability must be solely related to "woman with child" in the light of our experience with six male patients whose acute symptoms of phobia, dissociation, depression, and various degrees of psychotic withdrawal have occurred *only* and *recurrently* in close temporal relation to the pregnancy of their wives.

It is not implied that the analogous symptoms occurring in men or in women who have become ill following adoptions, abortions and miscarriages establish identical psychological mechanisms to those operating in the woman with full term child who develops para partum psychosis. However, these analogies do contribute to the concept that multiple factors unique in each individual may produce the illness.

The first victim of this point of view is the term, "para partum psychosis." Since there is a wide spectrum not only of effectors

but of the degree and direction of response to them, the dividing lines between "psychosis," and "delirium," and "neurosis," and "psychopathy" have become so blurred as to demand the substitution of the more accurate term "reaction" for "psychosis."

Survey of Statistics

In 51 of the 55 cases strong evidence of immaturity patterns was identified. Fifteen patients showed a predominant obsessive compulsive ritualism. There were fifteen patients with schizo-affective psychoses, six frank schizophrenias, and three manic depressive patients. One girl suffered a cerebral thrombosis one day post-partum followed by reactive depression and one for want of a better classification fell into the description of "acute toxic delirium."

Abstracting Chart II it will be noted that several of the major manifest symptoms and signs collected were as follows:

An expression of inadequacy in the care of the child	44
Guilt and self-condemnation	43
Severe depression, apathy and withdrawal	41
Rejection of the child	38
Slow reflex of identification with the child following childbirth	34
Suicidal impulse or attempt	34

Although 16 patients had various physical ailments attending their pregnancies, in only three were these considered enervating to such a degree as to interfere with adjustment to the maternal role.

In the list of family and hereditary factors 36 patients reported their childhood "unhappy." Thirty-two came from families in which their parents were separated, divorced, or severely maladjusted and on 31 the stamp of an inadequate father and a dominant rejecting mother made its mark.

In reviewing personality traits it is apparent that the most characteristic are referable to weak ego structure, in need of constant support from external sources:

Dependency patterns	49
Inferiority feelings	48
Chronic nervous tension	43
Inadequacy; clinging temperament	40
Passivity	38
Hysteria	35

Twenty-two presented a picture of childishness which was unmistakable. They were impetuous, narcissistic, babyish, whimsical, coquettish, credulous, and childlike in their associations. Thirty were obsessive-compulsive, perfectionistic, and rigid with a low tolerance for personal failure, falling into the anal erotic pattern.

Thirty-three had little experience with domesticity. Whether from over-protection by parents and siblings or early ineptitudes, or chronic illness in childhood and adolescence these girls had little familiarity with the responsibilities attendant on running a household or in the everyday care of infants and children.

A significant number, 24, had severe and unresolved affective and libidinal relations with their fathers in the classic "Electra" complex. The proposition by many analysts that the para partum illness is a reflection of penis envy is by no means universally found. Yet, for 19 repudiation of feminine goals, identification with masculine ideals, and the acknowledgment in deep insight therapy of their wish for the phallus was uncovered.

In general these women are unable to form firm love object relationships. They follow the pleasure principle, have a low tolerance to hostility in themselves, are narcissistic, manipulate others with childlike cunning and are pervaded by a feeling of inferiority and inadequacy to which the challenge of childbirth comes as a shock wave for which they are not prepared.

Of undetermined effect are aggregates of psychosocial factors catalysing in a society which has repudiated motherhood as a legitimate goal for feminine prestige. How much anxiety women have suffered from events of universal impact as economic depression and two wars can hardly be conjectured.

In evaluating situational factors it would be easy to assign crucial importance to these since they were prominent in the complaint of the patient and family, were quite easy to uncover and verify, and are generally acceptable as etiological vectors in all psychodynamic systems. However, in the factor analysis thus far completed, in no instance could it be said that the para partum reaction occurred from extrinsic pressures alone.

Thirty of the 55 cases divide the responsibility for their illness about equally among

familial, developmental, and situational items. In nine the familial and developmental factors seemed most important, in nine neuropathic traits plus severe situational stress aggravated disability, and in four severe trauma in a psychologically morbid childhood and adolescent environment erected a handicap to successful motherhood which even the most benign adult scene could not erase or compensate.

Among the situational difficulties the problem of housing loomed prominent, affecting 32 patients. These complained of having to live with their in-laws, of suffering from cramped space or of moving from one dwelling to another near the time of delivery. Twenty-six complained that their husbands were cold, inarticulate, unromantic and unsympathetic. Twenty-six were in severe financial difficulties for which they saw no ready solution. Twenty-five regretted their marriage to the wrong man and either admitted being in love with someone else or regularly phantasied erotic relations with previous boy-friends.

Twenty-three patients came home with their babies to a house ill-prepared to accept them. These women had little help from outside sources in putting together a pattern for life which would enable them to perform their domestic duties and give satisfactory care to their children.

A conflict with the family of the husband or wife was present in twenty instances to such a degree that the patient felt rejected and handicapped.

Discussion

A woman facing motherhood as a total threat to personality, sanity or life itself should probably be expected to suffer psychic collapse, just as the soldier who experiences danger, fatigue and constant deprivation may manifest combat exhaustion after his limit of endurance has been exceeded.[6] Yet this analogy is seldom recognized or acknowledged.

To many inadequate mothers pregnancy is a coalition of forces which threatens to destroy them. In defense of ego various patterns appear only one of which is the wish to destroy the child. Although 33 of the 55 patients rejected their infants with aversion only 20 had a strong thrust of homicidal feeling. *In more than half the cases, 35, the desire to annihilate the child was not the primary impulsion, recoil from which led to psychosis.*

Fear of picking up the child, lest it be dropped, of making a mistake in its formula lest it sicken, over-solicitude for minor ailments, constant obsessive watchfulness lest the baby smother in the blanket are frequently accepted almost as pathognomonic of masked desires to kill the child. They may be synonymous with hostility, but to imply that this is the invariable conclusion is unimaginative, dogmatic and misleading.

Frequently they signify the patient's conviction of her own insufficiency, ineptitude and worthlessness as a wife and mother. They may reflect her deep pessimism that nothing good can ever happen to her, or reactivate fears of deprivation as expressed by one young mother who said, "Everything nice I ever had was taken from me, everything I ever really wanted, I lost."

Even rejection of the child is a kind of magic ritual to prevent the dread event, *loss of the baby* from occurring.

It does not *pari passu* follow in every instance that because a patient has guilt and self condemnation as a prominent sign, that by that token alone homicidal desires be postulated. Guilt may be felt at a more fundamental layer of emotional reactivity. For her essential failure in the role of female; for the seeming validation of unconscious fears of homosexuality; for inability to face the crisis which childbirth (but not marriage) presents, namely the surrender of the father ideal and the acceptance of the husband as a love object. Many feel guilt because they see in themselves an instrument by which the neuropathic background from which they came will be perpetuated. They recognize in themselves neurotic needs for indulgence, affection and attention the solution of which has eluded them and which they well appreciate will be prejudicial to the welfare of their child.

These patients often know quite consciously that they are immature and that it is likely that they will reject their children as they themselves were rejected. The idea

of infanticide as a solution to the deeper problem hardly seems to explain the degree of guilt seen. In some instances the desire to kill the infant was a trigger setting off the flight reaction. In others it was not at all intense or critical. Fear of insanity, or loss of self control or of "doing wrong," as participation in homosexual or incest indulgences was more pertinent.

Desire and willingness to have the baby, yet fear of inadequacy in its care, and frustration of perfectionistic goals occurred in at least eleven patients in whom no trace of malevolence against the infant was found.

Proponents of the thesis that physical changes during delivery cause the para partum psychosis, as McGoogan,[7] Frumkes,[8] Ordway, and MacIntire,[9] Altschule,[10] Blumberg,[11] Frostig, Israel,[12] and Knaus,[13] may suggest that had we pursued biochemical studies of sufficient detail and exactitude we should probably find that hormonal unbalance is the vital etiologic factor in the cause of this illness.

Psychoanalysts as Zilboorg,[2,3] Freud,[14] Abraham,[15] Fenichel,[16] and Deutsch,[17] might insist that this study has not been intensive enough and might predict if analysis were continued according to strict Freudian protocol, that we should discover in each of the 190 cases that penis envy and castration fears aroused hostile homicidal drives against the baby leading to guilt, and flight into psychosis.

Since this progression was uncovered in about 20 patients of the 55 we cannot quarrel with it as one of several patterns influencing para partum morbid reactions, but that it is *the cause* of para partum psychosis, we doubt.

Any discussion of causation must begin with the appreciation that our criteria for what is causal is limited. Usually we use in one form or another the formal precept of Mills. In brief Mills states that whenever two phenomena are so related that the presence of one is necessarily conditioned by the presence of the other a causal relationship thereby exists, but it is disconcerting to reflect that an invariable antecedent or accompaniment of an event is not necessarily its cause and that our habit in science is to take one "invariable correlate" and label it "cause" when it is a factor that can be readily controlled or observed. Naturally the question of which antecedent will be called "the cause" depends upon the nature of the problem, and the point of view of the investigator.

What does emerge from this collation of data is that para partum psychosis is not a disease but an event in the life of the patient. One cannot diagnose history. One can only describe it as accurately and objectively as possible.

In the treatment of these patients supportive medical care was given in every instance and extensive psychotherapy conducted. Forty-one of the 55 patients had a series of electrocoma treatments of varying intensity prior to psychotherapy.

Results of Therapy

Patients who have experienced social recovery with good insight	12
Symptomatic recovery without relapse and good social adjustment	23
Recovery or improved, then relapse with subsequent improvement	16
Unchanged	2
Transferred to State Hospital	1
Suicide	1
Total	55

The tendency for this pattern to recur was seen in our first series. Fifteen of 18 primiparas and 16 of 20 multiparous patients suffered another severe mental reaction, usually symptomatically identical with their previous one, when they again became pregnant. None of these patients had been given psychiatric care in the prepartum periods.

Of twelve patients in this series of 55 who have become pregnant again and have been given prophylaxis, education, environmental manipulation, and in some instances endocrine preparations, only one suffered recurrence. On the other hand four patients from our study who have become pregnant in the last year and for one reason or another declined or could not continue with therapy have all relapsed.

This may indicate that a large number of para partum reactions may be prevented or attenuated. A careful examination of some of the factors within the control of the therapist in each individual case will reveal to

him the point to which his concentrated efforts should go to safeguard the patient from maternal psychological morbidity.

Summary

The case histories of 55 patients who have been intensively treated by one therapist are analyzed according to a breakdown of possible causative factors.

No aggregation of factors common to all patients was isolated.

The event of psychological illness paralleling the parturition period cannot be expected to have a universal causative agent because it is a result of multiple vectors of shifting force in the categories of hereditary, constitutional, physical, neuropathic, and environmental determinants.

Proper treatment for this condition will lead to a high ratio of recovery.

Prophylaxis is important in view of the high percentage (80 percent) of relapses in those who have had a previous psychotic reaction. This should consist of:

1. Search for techniques by which the patient's ego may be bolstered.
2. Re-education to the end that feminine goals may be found desirable and esteemed.
3. Development of insight as to the nature of the patient's reaction encouraged.
4. Control and limitation of stress factors in environment.
5. Discreet use of appropriate hormones as estrogen, thyroid and progesterone.
6. Adequate medical care and supervision of mother and child.
7. Prompt use of electrocoma therapy in the presence of impending suicidal and homicidal drives.

I

Syndromes

IMMATURITY	
Inadequacy	31
Depression and anxiety	24
Immaturity	19
Hysteria (conversion)	10
Passive dependency	7
Phobic reaction	6
Hypochondriasis	1
OBSESSIVE COMPULSIVE RITUALISM	15
SCHIZOPHRENIA	6
SCHIZO-AFFECTIVE PSYCHOSIS	15
MANIC DEPRESSIVE PSYCHOSIS	3
PSYCHOPATHY	1
LATENT & OVERT HOMOSEXUALITY	5
ORGANIC CEREBRAL DISEASE	1
DISSOCIATION SYNDROME	5
DEPERSONALIZATION	5
ACUTE TOXIC DELIRIUM	1

II

Manifest Symptoms and Signs

Expression of inadequacy in care of child	44
Guilt and self-condemnation	43
Depressed	41
Rejection of the child	38
Slow reflex of identification with the child	34
Suicidal impulse	34
Expression of obsessive ritualism	31
Expression of fear of insanity	26
Preoccupation with duty	21
Autism	21
Homicidal views against the baby	20
Delusions	20
Confusion	20
Exhaustion	18
Hallucinations	17
Dissociation	17
Homicidal views against parent, husband	14
Catatonic indifference and passivity	13
Phobic	13
Suicide attempt	12
Paranoid ideas	11
Conversion signs	11
Denial of the reality of the child	11
Hypochondriasis	10
Critical of child's development	7
Homicidal views in general	6
Manic reaction	5
Sexual promiscuity	3
Scoptophilia	1
Suicide, successful	1

III

Physical Complications Accompanying Parturition

Anemia	16
Vasomotor instability	16
Asthenia	11
Low BMR	7
Kidney disease	6
Malnutrition, avitaminosis	5
Miscarriage	5
Infection	4
Toxemia of pregnancy	3
Difficult labor	3
Hemorrhage	2
Cerebral thrombosis	2
Premature birth	2
Threatened miscarriage	2
Obesity	1
Hyperemesis gravidarum	1
Tubal pregnancy and abortion	1
Goiter	1
Epilepsy	1

IV

Family and Hereditary Factors

Unhappy childhood	36
Separated, divorced and maladjusted parents	32
Inadequate father and rejecting mother	31

Catholic	27
Rejecting mother	24
Emotionally unstable mother, domineering	22
Inadequate mother	21
Overprotective parents, siblings	20
Rejecting father	19
Early indoctrination in rigid moral standards	18
Sibling rivalry and striving	17
Strong religious affiliation with marked conflict	16
Severe premarital economic, social hardship	13
Suicide by a member of the family	10
Jewish	10
Protestant	9
Large family	7
Drunken father	6
Strict father	6
Menopausal psychosis in member of family	5
Stepfather	3
Stepmother	2
Tragic accidental death of member of the family	2
Psychopathy in family	1

V

Neuropathic Traits, Developmental Trends and Early Trauma

Dependency patterns	49
Inferiority	48
Chronic nervous tension	43
Inadequacy, clinging	40
Passivity	38
Hysteria	35
Little experience with domesticity	33
Hostility (poorly tolerated)	31
Obsessive compulsive	30
Attractive-feminine	28
Deep affective & libidinous relation with father	24
Attention getting mechanisms	23
Excessive shyness	23
Childlike associations (whimsical, impetuous, coquettish)	22
Emotional instability	20
Previous parapartum psychosis	19
Masculinized drives, penis envy, denial of feminine goals	19
Superiority-perfectionist strivings	18
Schizoid	17
Frigidity	17
Narcissism (pathological)	15
Guilt for sexual experiences	14
Phobic personality	13
Previous psychosis or mental illness	13
Aggressiveness	12
Masochism (pathological)	10
Illness, chronic in childhood	10
Dissociation symptoms	8
Guilt with regard to masturbation	8
Guilt for premarital, extramarital affairs	8
Jealous and possessive	6
Cyclothymic personality	5
Guilt re: violation of church dictum against use of contraceptives	5
Guilt re: promiscuity in past history	5
Organic inferiority: (Blemish, scar, deformity, obesity	4
Hypochondriasis	4
Premenstrual tension, severe	3
Homosexual experiences	3
Paranoid suspiciousness	3
Manic depressive psychosis	3
Alcoholism or drug addiction	2
Low I.Q.	2
Little or no experience in sex and love	1
Antisocial, psychopathic	1
Menopause	1
Serious accident, physical trauma	1
Great deal of overwork in early life	1
Severe sex trauma, premarital a) rape	1
b) abortion	1

VI

Situational Factors

Housing difficulties	32
Financial difficulties	26
Husband cold, inarticulate, unromantic	26
Patient regrets marriage to wrong man	25
Little help in pregnancy or post-natal period	23
Patient anxious and unhappy in pregnancy	22
Marital conflict and incompatibility	21
Parental rejection of patient and child	21
In-law trouble and family criticism	20
Disappointed in child (physique, ill, weight, sex)	16
Pregnancy unwanted	12
Happy in pregnancy	12
Fear of delivery	11
Sexual incompatibility in marriage	10
Loss of prestige in having child	10
Husband inadequate, maternal-bound, neurotic	10
Excessive responsibilities in care of home and child (objective)	10
Husband away (army, business)	9
Fear of pain	8
Obstetrician & hospital attendants unsympathetic, punitive	7
Pregnancy wanted and planned	7
Pregnancy unplanned	6
Husband rejection of patient and child	6
Length of stay in hospital postpartum too brief	5
Disappointment with delivery, sense of being "let down"	4
Pregnant too soon after marriage	4
Marriage in violation of church	4
Desired child for years unsuccessfully	3
Baby late in life (menopausal)	3
Acute illness of baby	3
Acute illness of patient	3
Four or more children previously "Family too big"	2
Separated	2
Divorced	2
Husband in extramarital affair	2
Difficult delivery, prolonged, caesarian, severe laceration, inadequate medical attention	2
Patient having extramarital affair	2
Psychosis in near-relation	2
Unmarried—raped	1

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Emotional Conflicts In Asthma

MILTON L. MILLER, M.D.
Beverly Hills, California

Ever since Hippocrates, it has been a question whether emotional factors play a role in precipitating asthma attacks. The copious earlier literature was summarized in detail by Dunbar[1] in her book on psychosomatic medicine. Wittkower[2] in 1935 and Alexander and French[3] in 1941 gave extensive treatment to the subject. In this last work, the research was done at the Chicago Institute for Psychoanalysis and constituted a turning point in the study of the influences of emotional factors in the causation of asthma attacks. It was conclusively demonstrated that emotional factors did play some role. The problem now was: What were the specific emotional factors involved, and how could they be explored?

The search for the more specific role of emotion in asthma remains a difficult and fascinating one. What was the basic conflict which most frequently contributed to the onset of an asthma attack? 27 cases, of whom 16 were adults, and 11 children, were studied psychoanalytically at the Chicago Institute. Their free associations, their dreams, and their most intimate personal activities, as told to their various analysts, were recorded during the analytic sessions, thus affording a massive body of material which could be studied quantitatively and comparatively The details of the patient's emotional state could then be precisely correlated with his changing allergic state; his actual attacks could be seen against the background of daily events, expressed or repressed emotions, dreams, and responses to psychotherapy. Of these cases 19 were under treatment for a prolonged period. The research patients varied widely in personality, intelligence, and social background, and ranged in age from small children to middle-aged persons. One specific conflict ran through every case. This conflict was related to the varied types of situations that precipitated the actual asthma attack. It was always a conflict about separation from the mother, fear of the loss of the protection and love of the mother or a mother substitute.

The patients in the study were all neurotic persons with asthma. If they had not had emotional conflicts they would not have been eager to undertake psychoanalytic treatment. Some had come to treatment primarily because of asthma and were referred by allergists, others came to obtain relief from problems of emotional maladjustment and were not aware that psychoanalysis might help their asthma. They differed from the general run of non-asthmatic, neurotic patients in treatment by psychoanalysis. Their personality make-up, at the deepest core, always betrayed the same weak point: Any situation that threatened estrangement from the mother or a substitute was cause for acute anxiety. They were typically ignorant and afraid about sexual maturity in their youth and grew up feeling that sexual temptation would deprive them of the much-needed maternal protection. Actual asthma attacks were brought on frequently by various types of sexual temptation. Actual physical removal from mother figures as in several cases of broken homes precipitated attacks. The birth of a younger child which threatened to estrange the patient from the mother, was at the root of the neuroses of quite a few. In some an attempt was made to overcome the threatened loss of the mother-child relationship by turning the situation around and mothering others. A number of successful careerists among the women either had a tendency to mother younger women, or to have homosexually tinged friendships on a mother-child basis. One patient, a delinquent girl, had homosexual relationships with several teachers towards whom she acted like a sulking, threatening child. The fear of the loss of the mother's love, created in some patients a submissive personality that of the good, obedient child. Others had a paradoxical reaction. By aggressive and provocative behavior, they caused by their own actions the very rejections they most feared. They provoked rejection from those they respected, rather than wait for the rejection they so deeply feared. Or else, they were afraid to establish a dependent relationship with anyone at all and withdrew from all, on whom they might be tempted to establish dependent ties. For example, one patient could never tolerate being told that her hour with the doctor was over, she always got up and left in advance.

It was found that these asthmatics were able to make use of an urge to confess what they considered their forbidden impulses in order to obtain forgiveness and reassurance which helped to ward off asthmatic attacks. Confessions were made to the analyst and also to parental figures. One young woman, for example, fell in love with a married man who was ready to divorce his wife and marry her. She revealed this situation to her own mother, who forbade her to see the man again, and the patient complied.

This therapeutic effect of confession to an understanding and forgiving figure to whom one can gradually unburden deeper conflicts is described by French and Johnson[4] in that paper, published in 1948. Insight into the relationship of the asthma attacks to the patient's sexual conflicts resulted in notably increased independence and freedom from these attacks. They quote a similar case described by Hansen[5] in 1930. The principle of reconciliation with a parental figure from whom estrangement was feared is put to practical use in the therapy of these cases.

To give an example: A man who had been suffering from asthma off and on for most of his life came to me to obtain relief from the asthma which had become much worse since his moving West. He had had to leave his hometown because of the severity of his illness. It was developed in interviews that his wife was a very difficult woman, frigid, hostile, and penny-pinching and he had been having an affair back home. He would come home late after being out with his mistress. He also used to gamble on horses. He was advised to move to a dryer climate. He retired from business and came to live in a different climate, but suddenly his asthma became worse again. He hesitatingly confessed in his interview that he had recently become attracted to a much younger woman, a friend of his niece, who had been visiting his house. He told his story expecting criticism, his eyes were downcast, he wheezed, coughed—but following the confession, there was marked improvement in breathing. After another interview or two, he was relieved of breathing difficulty for months. Many

months later, he returned, and confessed that he had begun to gamble on horses. This seemed a substitute for the interest in the girl, because he would, in discussing the horses, again refer to sexual thoughts and fantasies.

He was one of a large family and he had been the favorite of his mother. He confessed his indiscretions to me as if he were a boy confessing to his mother. The remarkable thing about him was that after confessing these impulses, he would get relief for weeks and months. The emotional component was reduced by these periods of confession.

The asthma attack itself appears to be the equivalent of a cry of repressed anxiety or rage. This was first described by Eduardo Weiss,[6] who compared the asthma patient to ". . . the shrieking, helplessly sprawling, newborn child with blood-red swollen face . . ." and likened the asthma attack to the anxiety resulting from the original separation from the mother, the birth experience.

Copious weeping occurs, very typically, with the emergence of anxiety connected with heterosexual impulses mobilized by analysis, or else when rage against the mother is about to be expressed during psychiatric interviews. It may be observed that an asthma attack sometimes gives way to a burst of sobbing, and as patients improve, they may have a crying spell as a substitute for the attack.

The findings of the Chicago Institute group are in accord with the work of three English researchers, Rogerson, Hardcastle, and Duguid,[7] who published a report of their study of asthmatic children at Guy's Hospital. They found the conflict over dependence on the mother so severe in these children, that they stated that most of the children would have found their way into treatment for nervousness, if not for their asthma.

In the actual psychoanalytic treatment of asthmatic patients, it has been learned that their relationship to the physician is very sensitive. Some have a tendency to lose the symptoms with little treatment, having gratified their wish to find someone on whom to be dependent. This type of cure, however, cannot be permanent. For lasting results, it is necessary to analyze the patient's defenses and so to resolve the conflict which is fundamentally responsible for the symptoms. Therefore, during psychoanalytic treatment, the symptoms may return temporarily in severe form. Many of the asthmatic patients treated at the Chicago Institute, reacted with relief or improvement of symptoms at the beginning of analytic treatment. Then when the analysis began to mobilize their fear of separation from the mother, the frequency and severity of the asthma increased temporarily. Later, if the analysis was carried to its conclusion successfully, there was a tendency for crying to replace the asthma, and in the end, if the personality matured, the need for marked dependence and the crying or asthma when this was frustated, began to disappear.

Margaret Gerard[8] adds data on five more cases of asthmatic children treated psychoanalytically. They all revealed conflicts similar to those found in the Chicago Institute study. Fear of separation from the mother stood out as the predominant theme. Along with increased independence, as a result of therapy, asthmatic attacks ceased completely in four of the children. The fifth child improved but had already developed secondary body changes evidenced in barrel chest. All five children were allergic to dust, some additionally to foods, pollens, etc. After analytic treatment, all five evidenced the same allergic reactions to tests, yet four of them ceased to have asthmatic symptoms.

Miller and Baruch,[9] in brief psychological studies, noted the strongly rejecting attitudes of the mothers of allergic children. Later in 1951 these authors reported a description of their findings in Abrahamson's book on Asthma.[10]

The question remains: How are allergic hypersensitivity and the emotional factors which apparently precipitate asthmatic attacks related to each other?

Abramson[10] describes the physiology as well as the psychology of asthma very thoroughly. He points out that the vagus and sympathetic interaction are disturbed in their innervation of the bronchi in asthma, but we really do not know why the bronchial spasm is utilized as an emotional expression. Suppressed crying for the mother, a regressive form of panic reaction, is the theoretical

explanation which has been advanced. The mechanism for the choice of the organ involved is not fully understood. Since the child's life depends on the close relationship to the mother's body, when this is broken, it is a severe trauma.

Dependence is orally expressed at first as in nursing. Later, gastro-intestinal symptoms are recognized as capable of expressing emotional conflicts of an oral type. But more recently, it has been noted that the respiratory tract, which happens to be embryologically of similar origin to the gastro-intestinal tract, and is also of importance in establishing early reactions to the outside world in the infant, is subject to the expression of conflict. The wish to stop breathing, to return to intrauterine life, the trauma of the infant at birth, being forced to breathe, breathing for the first time, is repeated in the neurotic use of the respiratory system.

Abramson also discusses Saul and Lyon's studies, in which Alexander and Saul's[11] findings that there is an individual pattern for breathing is quoted. Breathing patterns of an individual change somewhat during sleep. The expression of emotional conflicts in breathing plus allergic or other physiological circumstances have a complementary effect on each other, or more likely, there is a summation of effect. In some cases, the asthma may be purely physiological, in others, physiology works together with emotional factors. The emotional factors may lower the threshold of resistance to asthmatic attacks.

For example, a patient, a married man with two small children, was under psychoanalytic treatment. He thought his wife repulsive and claimed she was ugly. Actually, she was not ugly, and was rather intelligent. The patient's attitude was very immature. For instance, he would masturbate in bed, like a child. He had a mistress and did not attempt to hide the relationship. He was jealous of the mistress's interest in anyone else. His father had been prominent and the patient was an only child. He remembers his father had a mistress and the mother and father quarreled frequently over her. The mother was over-protective and remained so even when patient was a grown man. He repeated the pattern of his father in his life, and clearly equated his wife with his mother unconsciously. After he and his wife decided to separate he had his first asthma attack.

The general practitioner who deals with such a patient, particularly when the situation is temporary and acute, may find that he will respond favorably to an understanding listener, particularly when confession and weeping can afford relief. However, there are many asthmatic individuals who are unable to accept a dependent personal relationship toward a physician or anyone else, and they may take flight into health, *i.e.*, abandon their symptoms temporarily, only to suffer a severe attack later on, or they may have a provocative type of response: Fearing rejection if they establish any sort of dependent relationship, they may provoke the very rejection that they fear, by resentful, aggressive behavior. Or else they may build up a dependence on the physician that is so great that they react with illness to his vacations, or curtailment of their visits to him. The asthmatic is an anxiety-ridden individual. Particularly, one must be cautious about interpreting his basic conflicts about relationships to mother figures—one cannot simply say, "You have an attack of asthma because you are afraid to leave your wife, she represents your mother to you." Such a statement may produce no effect at all—or else produce profound anxiety and a great deal of acting-out.

Summary

The extent of the role that psychological factors play in asthma has been dealt with copiously in medical literature. Recent research has begun to explore the specific nature of emotional factors which, by complementary action or summation, relate themselves to allergic factors causing asthmatic attacks. Outstanding contributions to this problem in the last decade have brought out the following: The asthmatic in whom neurotic factors play a prominent part has been found to suffer from an unsolved conflict over dependence upon the mother or mother substitute. The asthmatic attack is like a suppressed infantile cry for maternal aid and protection. This basic emotional situation occurs in greatly varied

types of individuals and is overlayed with numerous and often paradoxical defense reactions. Many ranges of intelligence, age, character, and social background occur, yet the uniformity of the specific emotional conflict at the core has been striking, in the psychiatric experience thus far accumulated. The relief obtained by confession, by crying, and by the solution of acute conflicts over separation from maternal persons in the patient's environment has had beneficial effects.

It remains for future research to clarify the physiological mechanism by which the innervation of the bronchi is disturbed when the patient's allergic condition and emotional conflict co-exist in the causation of asthmatic attacks.

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Cerebral Biopsy In Patients With Presenile Dementia

MARTIN A. GREEN, M.D., LEWIS D. STEVENSON, M.D., JUAN E. FONSECA, M.D.
and S. BERNARD WORTIS, M.D.
New York, New York

INTRODUCTION

In discussions of patients with presenile dementia seen in Bellevue Hospital the question always arises as to whether such patients have Alzheimer's disease, one of the more well known types of presenile dementias. The prevailing opinion has been that this condition can be differentiated from other primary presenile dementias only on the basis of the characteristic pathologic alterations in the cerebral cortex and not on the basis of its clinical features. The present study was undertaken to determine whether such a view is warranted or whether Alzheimer's disease does form a specific clinical entity distinguishable from other primary presenile dementias.

SUBJECTS AND METHOD

Nineteen patients were studied. In 15 cases the clinical diagnosis was primary presenile dementia. These patients showed gradual and progressive intellectual impairment and behavioral changes in the presenile period (40-60 years of age) for which no etiology could be found on neurological study. In the remaining 4 cases the clinical diagnosis was cerebral degenerative disease, cause unknown. Although these patients also showed dementia in the presenile period, the onset and course of their illness was different from that usually seen in presenile dementia and hence they were separated from the preceding group. This difference will be discussed later in the paper.

Each patient had a lumbar puncture, skull x-rays, electroencephalogram, psychometrics (Wechsler-Bellevue Intelligence Scale and Bender Gestalt), and either pneumoencephalography or ventriculography.

The biopsy was obtained by use of a one-inch trephine in the right frontal region the skin was usually opened as a small flap, preferably behind the hairline. A small dural flap which could later be closed gave the best exposure and allowed better choice of an avascular area. A circle corresponding in size to the inner diameter of the glass tube used to aspirate the biopsy was then lightly cut in the arachnoid and pia with a #11 blade. The aspirating tube was an ordinary hospital drinking tube made of glass approximately 0.5 cm. inside diameter. One end of it was applied to the circle incised on the brain and a 10 cc. syringe was attached by a rubber connection to the other end. The tube was then gently pushed in with a slight rotary motion to a depth of one inch; as gentle suction was applied, the cylindrical specimen could be seen to remain in the tube as if punched out. When the adequate depth was reached the suction was increased somewhat as the tube was withdrawn. Occasional bleeding could usually be controlled with a small gel foam pack. The tissue was stained by the following methods: Mallory's trichrome, hemaoxylin and eosin, toluidine blue, Weil's mylin sheath stain, Sudan IV, Cajal's silver nitrate for neurofibrils, and Hortega's silver carbonate for astrocytes and microglia.

The criteria for the diagnosis of Alzheimer's disease from the biopsy were the presence of Alzheimer's cells, senile plaques, diffuse gliosis, and an abnormal amount of fatty pigment in the nerve cells. Biopsies showing non-specific degenerative nerve cell changes, abnormal accumulations of fatty pigment, and gliosis but without Alzheimer's cells, senile plaques, or evidence of inflammatory or vascular disease were diagnosed as encephalopathy, type undetermined.

RESULTS

Patients with Alzheimer's Disease:

The biopsies in 7 of the 15 cases of presenile dementia showed alterations indicative of Alzheimer's disease. The duration of symptoms for 2 of these patients was 1-2 years and for the remaining patients 3-5 years. In every case the initial and out-

From the Psychiatric Division of Bellevue Hospital and the Departments of Neurology, Pathology, Surgery and Psychiatry, New York University College of Medicine.

standing symptom in the development of the illness was progressive intellectual deterioration and alterations in behavior rather than focal neurological signs. There was no characteristic pattern in either the intellectual impairment or the behavioral disturbances. Disorientation, memory impairment, defects in calculation and general information, perseveration, emotional lability, and concreteness in thinking were present in varying degrees and combinations. Elements of aphasia and apraxia were intermixed with the general intellectual impairment in most instances and in two cases the aphasia was so marked that little contact with the patient was possible. Only one patient had focal neurological signs, which consisted of a left homonymous hemianopsia and a left hemisensory defect. Some patients were voluble while others were underproductive. This was unrelated to the severity of the aphasia. Four patients were quiet and cooperative while two patients were agitated and hostile and had to be put into restraints intermittently. Two patients showed pronounced emotional lability. All but one patient was incontinent.

On psychometric examination the intellectual defects seemed all pervasive and affected every area of test functioning. There was no characteristic pattern in the response which would distinguish these patients from patients with organic brain disease other than Alzheimer's disease. The Bender Gestalt reproductions were characterized by perseveration, dissociation of figures, difficulties in angulation and maintenance of curvature, regressive-deteriorative features, disorganized approach and planning, and rotation of figures. The Wechsler-Bellevue showed markedly lowered I.Q.'s with great losses in all areas but most consistently present on block design, digit span, and arithmetic problems. In all cases thinking was highly concrete and bound to the demands of the immediate situation.

Lumbar puncture showed normal spinal fluid findings in all but one case (Case #4). In this case there was persistent pleocytosis and an elevated total protein. The Wassermann reaction and virus studies of the spinal fluid were negative. The etiology of the spinal fluid abnormalities was not determined. The clinical history was not indicative of inflammatory disease and the cerebral biopsy did not show inflammatory alterations.

Electroencephalogram was obtained in 5 cases. No specific pattern characteristic for Alzheimer's disease was found. The records showed symmetrical slowing of alpha activity and very slow delta waves (large, irregular 1½-3 per second), diffusely, but most marked in the temporal and posterior parietal regions with greater involvement on one side of the brain.

Cerebral atrophy was demonstrated by pneumoencephalography in every case. This was manifested by dilatation of the ventricular system and increased cortical markings. There was a wide variation in the severity and extent of the atrophy but in all instances it appeared diffuse rather than focal.

A summary of the cases follows:

Case No. 1: A 61 year old male had a four year history of progressive memory impairment and general intellectual deterioration. Examination showed a severe organic mental syndrome with elements of aphasia. Speech was underproductive and difficult to understand. Apraxia, ecolalia, and perseveration were present. He reacted only slightly to painful stimuli. There were no focal neurological signs. Most of the time the patient paced about the ward talking to himself. Later he became more restless, annoyed other patients, and had to be restrained. A cerebral biopsy was taken from the right frontal region.

Case No. 2: A 58 year old male had stopped speaking English 1½ years previously and spoke only Italian, his native language. He had headaches, memory defects, and abdominal distress for one year. Six to seven months before admission he stopped playing the piano, a favorite recreation, and speech in Italian became unintelligible. He had several "fainting spells" but no convulsions. He had received electric shock therapy just prior to admission and was said to have shown some improvement. On examination his speech was an unintelligible jargon in Italian and he was unable to follow commands given in Italian. He could neither speak nor understand English. There were no neurological signs. He became agitated several times during his hospital stay and had to be transferred to the disturbed ward. He was incontinent and unable to feed himself. A cerebral biopsy was taken from the right frontal region.

Case No. 3: A 45 year old female had a 3 year history of gradual loss of the ability to calculate and episodes of confusion during which she misidentified her husband and expressed paranoid ideas. Headaches, nosebleeds, and abdominal pains which

she had had at least 16 years became more severe. She thought she had cancer and was treated by numerous physicians and chiropractors. She described attacks of falling or fainting although her husband never observed them. She continually threatened suicide and actually made two attempts to hang herself. General physical examination was negative. Blood pressure was 105/65. On neurological examination there were defects in all spheres of the sensorium but there was a particularly severe loss in recent memory and in retention. There was no aphasia or apraxia. There were no focal neurological signs. She spoke readily but in a disconnected manner. She expressed somatic delusions and was extremely hostile toward her husband. There were rapid changes in mood. At times she appeared depressed and at other times euphoric. She laughed and cried intermittently. She was cooperative in the ward routine and able to feed herself and was not incontinent. No convulsions or fainting spells were observed. A trial on Tridione did not alter her mental status. A cerebral biopsy was taken from the right frontal area.

Case No. 4: A 47 year old woman began to nag and holler at her family and lost interest in housework 3 years previously. These behavioral changes became progressively more severe. Six months before admission she was unable to dress herself and later became incontinent and forgot her childrens' names. On examination very little contact with the patient could be established. There was no spontaneous speech and there was a marked paucity of productions on questioning. She was friendly and smiled pleasantly but only occasionally would she say a few words in response to questions. Sometimes she responded in a totally irrelevant manner with a Yiddish blessing. She was unable to name simple objects. She did not obey simple commands, imitate, or respond to the handshake gesture. There were no focal neurological signs. She was cooperative and able to feed herself. She was incontinent of both urine and feces. A cerebral biopsy was taken from the right frontal lobe.

Case No. 5: A 50 year old male had a 3 year history of progressive impairment of memory. During the several months prior to admission he had become confused and wandered away from home at times. The only neurological signs were severe sensorial defects and a marked aphasia. There was little spontaneous speech. Spontaneous crying was present. The patient was quiet, acquiescent, and pleasant. He wandered about the ward in a confused fashion, staring vacuously into space, and was obviously unable to comprehend his situation. He was incontinent. A biopsy was taken from the right frontal region.

Case No. 6: A 58 year old woman had a 4-5 year history of mental and personality changes. Initially she began to mislay objects and complained of occasional headaches. During the next 2 years she repeated herself in conversation, did not play bridge as often as before, and complained of a constant feeling of fatigue. Later she had difficulty in making decisions, became careless in dress, and exhibited stereotyped movements when excited. Subsequently she became confused and incontinent. Neurological examination disclosed an organic mental syndrome with aphasia and apraxia. There was a left homonymous hemianopsia and a left hemisensory defect to pin prick and touch. Generally the patient was friendly and cooperative, although sometimes she became depressed and cried. This occurred particularly in test situations. A cerebral biopsy was taken from the left parietal lobe.

Case No. 7: A 60 year old male had a one year history of progressive memory impairment, difficulty in concentration, and mood disturbances. He had episodes of confusion and would lapse into irrelevant speech. He became querulous and depressed and cried frequently. He spent many hours talking to himself. On examination there was an organic mental syndrome with elements of aphasia and apraxia. He was friendly and euphoric. He was frequently seen standing before a mirror talking and gesturing to his image. When asked about this he maintained a persistent delusion that the image was that of his brother. A cerebral biopsy was taken from the right frontal region.

Patients with Biopsies Showing Encephalopathy, Type Undetermined:

The cerebral biopsies from 8 patients with presenile dementia showed encephalopathy of an undetermined type, except in one instance in which the changes resembled those seen in Pick's disease. Clinically these patients were indistinguishable from those with biopsies showing Alzheimer's disease. The typical history was that of progressive intellectual impairment and behavioral changes. Elements of aphasia were sometimes present along with the organic sensorial changes presented by each patient. There were usually no focal neurological signs. Considerable variation in speech production, affect, and activity was noted as in patients with Alzheimer's disease and no general pattern was evident. The responses on psychometrics showed features similar to those shown by the patients with Alzheimer's disease. The spinal fluid was normal and air studies usually demonstrated diffuse cerebral atrophy. Electroencephalography showed generalized slowing of alpha activity plus focal abnormalities consisting of voltage depressions or paroxysmal activity not confined to any particular area. Because of these features the records less specifically suggested a diffuse encephalopathy than did those from the patients with Alzheimer's disease.

Two patients had mild hypertension, in one associated with auricular fibrillation. The history in both instances was that of progressive dementia. There were no episodic attacks of nervous system dysfunction that would suggest cerebral vascular disease. The biopsies from both patients showed thickening of the walls of the arterioles and a few perivascular hemorrhages.

Patients with Cerebral Degenerative Disease:

The biopsies of the 4 patients in this group all showed encephalopathy of an undetermined type similar to the proceeding group. As stated previously although the onset of dementia in these patients occurred in the presenium, the development of the illness was different from that usually seen in the primary presenile dementias. For this reason the diagnosis was cerebral degenerate disease, cause unknown rather than the more specific diagnosis of presenile dementia. In two cases the onset of the organic mental changes occurred suddenly just prior to admission. Another patient had an 8 year history of focal and generalized seizures and progressive neurological signs indicative of diffuse disease of the cerebral hemispheres. Some of the neurological disability was apparently the result of a meningitis following a ventriculogram done during the course of his illness. The history in the last case was inadequate. For at least 20 years the patient had been shy and eccentric, lived as a recluse, and had had auditory hallucinations. Examination showed an organic mental syndrome, right lower facial weakness, and diminished arm swing on the left. The diagnosis was cerebral degenerate disease superimposed on a previous long standing schizophrenia. In each of the four cases neurological study failed to disclose the etiology of the encephalopathy.

Morbidity and Mortality from Biopsy:

No neurological disability developed after biopsy that could be attributed to the operation. There was one instance of post-operative hemorrhage, and infection, in the form of a small stitch abscess, occurred in another case. Three patients died but none of these deaths could be related to the effects of the operation. Two of these patients died from head trauma sustained during hospitalization and one patient from what was probably coronary thrombosis.

DISCUSSION

Different opinions have been expressed in the literature as to whether Alzheimer's disease is a distinct clinical entity that can be differentiated from other primary presenile dementias, principally Pick's disease.[1-6] The prevailing opinion is that such differentiation is difficult if at all possible and that these conditions can only be identified on post-mortem examination of the brain. It is felt that the variation in the clinical manifestations of these diseases is considerable and that no one symptom or sign is characteristic. In general, however, patients with Alzheimer's disease as contrasted to those with Pick's disease are described as showing earlier and severer memory disturbances, hyperactivity, diffuse rather than focal neurological signs, and frequently seizures. Patients with Pick's disease are described as having less severe memory defects which appear later in the course of the illness, being apathetic and inactive, having focal rather than diffuse neurological signs, and only rarely having seizures. A family history of presenile dementia and the presence of symmetrical focal atrophy on pneumoencephalography are given as additional factors favoring the diagnosis of Pick's disease.

Although the clinical observations in the present study were not as complete as previous studies, the findings support the view that Alzheimer's disease does not form a distinct clinical entity. In most of the patients the signs of the disease had been present for over three years, a relatively long period. Despite this fact exceptions to the supposed characteristic features of the disease mentioned previously occurred. Some patients were quiet, apathetic, or depressed rather than hyperactive. None of the patients had seizures. However, the absence of focal neurological signs as a feature of the disease held true for all but one patient and in general the memory defects were severe. A familial history and the pneumoencephalogram were not helpful since an adequate family history was not obtained in most instances and air studies invariably showed diffuse cerebral atrophy.

The only generalization that can be made concerning the clinical syndrome in patients with Alzheimer's disease in this study is that the disease was manifested by a progressive organic dementia without focal neurological signs. There was no characteristic pattern to the organic psychosis, either clinically or on psychometric examination, nor was there any pattern of behavior common to a majority of the patients. Similarly, laboratory tests, such as lumbar puncture, pneumoencephalogram, and electroencephalography, failed to show findings specific for the group.

The patients with primary presenile dementia with biopsies showing a non-specific encephalopathy rather than Alzheimer's disease also failed to form a uniform clinical picture and were indistinguishable from the patients with Alzheimer's disease. It is of course realized that some of these patients may have had Alzheimer's disease, the characteristic pathological alterations being absent from the small sampling of cerebral tissue that was obtained. Unfortunately no autopsies were secured in this group. However, considering the severity and omnipresence of Alzheimer cells and senile plaques in Alzheimer's disease it is difficult to imagine that they would be entirely absent in even a small piece of tissue such as a biopsy. It is felt that at least most of the patients in this group had a presenile process other than Alzheimer's disease. Just what this might have been is conjecture. Possibly some might have had Pick's disease but in only one biopsy did the nerve cell changes bear any resemblance to those seen in this disease. Some of the cases may have been one of the more obscure presenile encephalopathies. Bender, Wortis, and Cramer[7] described such a case thought to be Alzheimer's disease but which showed a diffuse demyelinating process on post-mortem examination. The point to be emphasized is that in a group of patients with primary presenile dementia it was not possible to distinguish clinically those with Alzheimer's disease from those with other types of encephalopathies.

This study also indicates that Alzheimer's disease may be more frequent than commonly thought. Approximately 150 cases have been reported in the literature. Rothchild and Kasanin[2] found a 4 per cent incident in a series of 234 necropsies and McMenemey[8] reported 1.2 per cent of cases in a series of 487 consecutive autopsies, both studies being done at a mental hospital. In the present study carried out over a year's period, 7 out of 15 patients with primary presenile dementias, approximately 50 per cent, showed Alzheimer's disease. This does not represent the maximum number of such cases admitted to the hospital. Additional similar patients could not be included because of the failure to obtain consent from the family for biopsy.

SUMMARY

Cerebral biopsies done in 15 patients with primary presenile dementia showed Alzheimer's disease in 7 cases and a non-specific encephalopathy in 8 cases. The findings on clinical examination and laboratory tests were similar in both groups of patients. It is concluded that Alzheimer's disease cannot be distinguished clinically from other types of primary presenile dementias. Such differentiation is possible only on pathologic examination of the brain.

Authors' Note: The authors acknowledge with thanks the assistance of Miss Esther Stearns, of our staff, in interpreting the EEG's.

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Neurological Clinical Pathological Conference

CHARLES D. ARING, M.D., *Editor*

CASE NO. 165143

Presentation

A 48-year-old white man entered the hospital for the second time on October 6, 1951 for treatment of burns received eight days previously. He died January 13, 1952. Born of Mexican parents in Houston, Texas, he was orphaned in early childhood and had never gone to school. As a child he tended donkeys. As he grew up he did hard labor in various parts of the country. He visited in Mexico in 1925. He came to Cincinnati in 1926 and married a woman 5 years his junior. The marriage was happy but she had never become pregnant.

His health was good until August 1940, when he developed severe occipital headaches. About a year later his legs became weak, causing him to fall on several occasions. There had developed festination and propulsive gait. There were brief periods of mental confusion which accompanied the transient paralysis of his legs. He had fallen several times because of this weakness. Remissions and relapses were characteristic of his difficulties. On October 15, 1941, he was found wandering on the street in confusion and was brought to the hospital by the police.

He was confused and could not give a coherent story. He said, "I don't know why I am here." He also had language difficulty. On one occasion he said that he had received injections after a penetrating wound of the foot. His wife had no knowledge of any antisyphilitic treatment.

The general physical examination was normal. Cranial nerves were entirely normal. It may be worth noting that examinations of the optic fundi and pupils were thorough and they were completely normal. Active deep and skin reflexes were elicited throughout. The plantar responses were extensor bilaterally and sustained ankle clonus was present on the left. Despite his complaint of weakness in the legs, the first examination revealed no loss of strength. During his stay on the ward repeated neurological examinations showed minimal weakness in the left limbs. He walked with a broad-based shuffling gait, with the body bent slightly forward from the hips. Associated movements were normal. Finger-to-nose test was normal and there was no past pointing. He showed tremor of the legs on the heel-to-knee test. No abnormalities of sensation were elicited on careful testing.

Blood counts and urinalysis were normal; the blood pressure was 120/80. A Kahn test of the blood was negative. Lumbar puncture on admission yielded clear fluid with initial pressure of 310 mm. of water and final pressure 240 after removal of 10 cc. The fluid contained 43 lymphocytes per cmm. Wassermann was negative on the cerebrospinal fluid. The total protein was 33 mg. per cent. Repeated lumbar puncture 5 days later showed an initial pressure of 280 mm. of water and final pressure 165 after removal of 10 cc. The cell count was 43 lymphocytes and protein 40 mg. Wassermann was again negative and the gold curve was 4433210000. An electroencephalogram showed abnormalities in the right frontal and parietal regions and less obvious changes in the same areas on the left.

His general conduct on the ward was that of withdrawal and confusion, but he was cooperative. During the first week he had fever to 100.8° but was afebrile thereafter. He seemed disinterested in ward activities, was quiet, sometimes did not answer questions. He was slow to comprehend, occasionally he urinated in bed. He had urinary urgency. His movements were very slow, his facial expression blank. Throughout his hospital stay he seemed dazed, would sit off by himself staring at the group activity, never seeming to comprehend what was going on or why he was here.

He had been in an extremely filthy condition on admission to the hospital. He was illiterate although his wife had taught him to write his name. Thorough mental testing was done. His answers were very slow, rather facetious, and never consistent. His memory was affected generally; he could do simple calculation except subtraction. He never seemed to be able to derive the meaning from conversation; this "fog" remained except for moments of relative lucidity through his sixteen days in the hospital. He was given one intramuscular injection of bismuth and discharged October 30, 1941, with an appointment for the Venereal Disease Clinic.

He did not attend the clinics of this hospital but received some anti-luetic treatment from a private physician. There was transitory improvement but he did not completely recover from his illness and was never able to resume heavy work. He and his wife lived in a second floor apartment and for the last two years of his life, walking was so difficult that he was unable to leave home. In the last 6 months of this period he became considerably worse, found increasing difficulty in walking, and fell with increasing frequency.

On September 28, 1951, while his wife was at work and he was alone at home, he fell while attempting to pour a cup of coffee and was burned. A physician treated him at home but referred him to the hospital 8 days later when the burns became infected. He was confused throughout this hospital stay. Ophthalmoscopic examination was normal. The pupils reacted sluggishly to light. While there was some generalized weakness, it was most obvious in the right limbs. Deep reflexes were hyperactive in the lower extremities. The abdominal skin reflexes were absent, and there were extensor plantar responses bilaterally. Third degree, infected burns were present over the posterior thorax and right arm.

On admission the temperature was 100°F., pulse rate 94, respiration 20, and blood pressure 180/110. Hemoglobin was 12 gm. per cent and the white cell count 7,000. The urine was normal except for an occasional white cell in the sediment. A Kahn test of the blood was negative. Serum proteins were 8 gm. per cent and the A/G ratio 1.8:1. Lumbar puncture was not performed because of the infected burns and decubital ulceration. On October 25, 1951, an electrocardiogram showed low voltage of T waves in all leads.

Date	*BUN*	*Serum Bilirubin*	*CO_2*	*Cl.*
10-21	32 mg.%	2.7/4.3 mg.%	18 meq/1.	93 meq/1.
10-22	54		17	93
10-23	57	1.7/2.7	18	99
10-24	54		17	97
10-26	41		13	108
11-21	7		20	95
11-30	16		17	92

A series of portable chest films showed: October 20, no significant abnormality; October 23, suggestive atelectasis left lower lobe; October 25, atelectasis of left lower lobe, slight mediastinal shift and distended stomach; October 28, atelectasis left lower lobe; October 30, no mediastinal shift, obliteration of left costophrenic angle by fluid and some infiltrate in left lower lobe. PA and left lateral teleoroentgenograms of the chest November 6 showed a wedge-shaped density in the posterior part of the left lower thorax. The lung bases showed disc atelectasis and the cardiac shadow appeared normal. Chest films were repeated November 15 and showed no change on the left but a hazy density in the right lower thorax. Portable films of the abdomen January 9, 1952, were unsatisfactory but showed distention of stomach and colon.

He was involuntary of stool and urine. On October 9 there were several convulsions; one that was observed began on the right side and left him with more weakness of the right limbs than previously. He was given penicillin, dilantin, and phenobarbital, and the burns were treated with Dakin's solution. Temperature of 101° during the first week fell to 99° during the second. Penicillin was discontinued and his course was satisfactory with healing of the burns until October 19 when he was found cyanotic and in shock. A friction rub was heard over the lingula and P_2 was greater than A_2. He was given oxygen and anticoagulants but continued to have dyspnea and cyanosis. Anticoagulant therapy was discontinued and blood administered October 22 when gross hematuria occurred. Cyanosis and dyspnea continued and copious mucopurulent material was aspirated from the left main stem bronchus by bronchoscopy which was done twice. Tracheotomy was performed October 26 in order to achieve more effective suction of the bronchial tree. Fever to 104° returned during this period and penicillin and chloromycetin were given. He then improved slightly but developed decubiti and became a feeding problem. Tube feedings were not tolerated and jejunostomy was done November 26. With temperature around 99.6°, antibiotics were discontinued December 8. There was little change in his condition until December 25 when he again had fever to 104°, rales were heard in both lungs, the urine was loaded with pus cells and yielded E. coli on culture. Chloromycetin and gantrisin were given. High fever continued and he had drenching sweats. He died January 13, 1952.

Differential Diagnosis

Dr. Joseph P. Evans: The problem for today is that of a 48-year-old man who had two hospital admissions, the first in 1941 and the second ten years later. In the earlier history we learn that this Mexican, at least born of Mexican parents in Houston, attended donkeys as a child. I suspect there must be some significance to this observation. Then, after he grew up, he went to Mexico and then came to Cincinnati where he was happily married though he had no children. This is a matter of some importance in relation to the obvious diagnosis that was made at the time of his first admission.

In 1940 he began having difficulty with headache about which we apparently have little information, but in 1941 he was brought into the hospital because of confusion, having been picked up by the police on the street. At that time the salient points in his examination were the mental confusion, pyramidal tract signs and a broadbased gait, findings suggesting rather diffuse involvement of the central nervous system. Laboratory tests, save those on the cerebrospinal fluid, were essentially normal and it is worth noting that the blood Kahn was negative. Lumbar puncture pressure was recorded as 310 mm. of water on one occasion and 280 on another, so I think that we may presume that he had increased intracranial pressure. There was an increased number of cells, 43 cells on two occasions, in the cerebrospinal fluid, all of them lymphocytes. Total protein determinations were 33 mg. and 40 mg. per cent. Electroencephalograms showed diffuse abnormalities.

Now I should think that it is quite evident from the fact that he was referred to the Venereal Disease Clinic, that the diagnostic conclusion at that time was that he was suffering from central nervous system lues. I presume they were thinking of paresis. In retrospect, I think there is reason to doubt the validity of that diagnosis, and on the information which we have it would really be difficult to defend that initial diagnosis.

Apparently matters improved for a time, but obviously he did not recover health because he was not able to resume heavy work, and in the final two years of his life he was unable to leave his second floor apartment. The incident on September 28, 1951, when he spilled the coffee, suggests that he must have had some degree of ataxia. It is difficult to understand why he should have spilled the coffee in this fashion unless he was very ataxic. He was brought to the hospital, because of infections from these burns, 8 days after the incident. I think most of the subsequent course, though it was lethal in nature, was incidental to the problem at hand. On this admission he again showed evidence of pyramidal tract disturbance, incoordination, and furthermore had become incontinent. His mental status was not commented on, but I would judge that there was at least some fogginess because of the vagueness of the interval history. There is also

the record of hypertension on this admission, a development since 1941 when the blood pressure was 120/80. Then we note that on the third hospital day he had several convulsions, followed by right-sided weakness. In the record of the hospital visit ten years earlier there had been some comment about left-sided weakness.

Matters seemed to be going along reasonably well until October 19 when he became cyanotic and went into shock. The only reasonable conclusion that I can draw from this is that he had suffered a pulmonary infarct. The physical findings which were recorded immediately thereafter are confirmatory of this.

I think the laboratory studies can be related to the course of his illness. The anticoagulant therapy apparently was pushed vigorously. He developed hematuria, and I suppose that it is reasonable to conclude that he may also have had some melena at that time, which may account for the increased blood urea nitrogen. The serum bilirubin gives some evidence of mild hepatic damage indicating intrahepatic jaundice. The explanation of the diminished CO_2 is not clear unless perhaps he had hypernea in association with the overall picture, nor can I account satisfactorily for the slight reduction in the chlorides. I think at this point a discussion of the roentgen studies is in order.

Dr. Emanuel Levin: The film taken at the time of admission showed a cardiovascular silhouette of normal size. There was no evidence of aneurysm. In a series of portable films there were some slight respiratory excursions, so that the diaphragm was blurred. A few days later the heart had been displaced to the left hemithorax, there was a hazy density in this area and probably some gross dilatation of the stomach with displacement of the heart, and there was some collapse of the chest. The left hemithorax was smaller in volume than the right, signifying atelectasis of the left lower lobe.

In a later film the atelectasis was still present with probably a slight degree of pleural effusion. At that time there was a tracheotomy tube in place. On November 6, 1951, there was a considerable degree of clearing so that the heart was again in about normal position. There was only some disc atelectasis in the left costophrenic sinus and the size of each hemithorax was about equal. A lateral film taken at the same time, however, showed triangle density far posteriorly at the base which demonstrated the collapse of the lower lobe.

A posterior-anterior teleoroentgenogram taken on December 12, showed a hazy density at the right base which probably represented an early pneumonia. The increased density behind the left heart was still present showing that there was still atelectasis of the left lower lobe. A film of the abdomen taken a few days prior to death showed gross distention of the stomach, the small and large bowel, and a picture of a paralytic ileus. These films give us no hint of the reason.

Dr. Evans: I would presume that these pulmonary difficulties are reflections of atelectasis secondary to the infarction. While one would like to relate them to the primary difficulty, I think that it is difficult to do so with any degree of assurance. This same would hold for the paralytic ileus which was demonstrated in the last film.

We are left then with evidence of diffuse, long-standing central nervous system involvement as indicated by the confusion, by the long tract signs, the incoordination and the incontinence; also by the occurrence of convulsions and weakness first noted on the left side of the body and ten years later on the right side. There was electroencephalographic evidence on the first admission of change, rather diffuse, and there were the cerebrospinal fluid findings which we have already noted, indicative of increased intracranial pressure, and of some type of process which would call forth a lymphocytic response. Furthermore, we have evidence of liver disease which may be related to the primary process. The pulmonary infarction may only have been a reflection of his general debility and been phlebo-thrombosis or possibly thrombophlebitis, though I think one must consider, too, whether this is related to the primary difficulty. The evidence of blood vessel disease, and the low voltage T waves and hypertension would suggest at least mild cardiac hypertrophy.

In any neurological diagnosis, one attempts to make a localization which, however, in this instance is difficult. There is evidence of diffuse disease affecting the cerebrum and probably the cerebellum as well. There is suggestive evidence that the cord may have been disturbed in addition.

Congenital or developmental disease may be eliminated because the man was apparenly well until some 10 or 11 years before he entered the hospital. There is no history of trauma. We shall have to consider infectious processes. I don't believe we have to consider seriously metabolic or toxic diseases. We certainly have to think about vascular disease and on his first admission this possibility was seriously considered, a combination of vascular and infectious, in other words lues. We do have to consider the possibility of neoplasm. In view of the general nature of this picture I think that we do not have to consider too seriously the matter of degenerative disease.

Now in considering the etiologic possibilities further, a possible answer might be carcinomatosis or sarcomatosis of the meninges. However, these symptoms are of rather long duration for such a process. But I recall that we have a female patient with sarcomatosis of the meninges that we have been following since 1943. She was operated upon at that time and has a confirmed diagnosis of sarcomatosis. With x-ray radiation she has gone through this period of years without being in too serious trouble. So that certainly it is a possibility which ought to be considered. That is, I think, the only neoplastic process that might conceivably cause this diffuse lesion and have been of such long standing. From the standpoint of vascular disease, lues is the obvious process one would consider, but I would feel that that has been ruled out by the initial findings and also by the subsequent course.

Then one is left with the consideration of what infectious processes might be responsible for this disturbance. I hardly think that any of the ordinary agents with which we deal, bacterial agents, could be considered a factor here. There remains, then, the possibility of low-grade reactive processes such as yeasts or parasites, and I would like to read from Bailey's textbook on *Intracranial Tumors* because it seems to me that perhaps this recapitulates our problem:

"In this country parasites do not often enter into a differential diagnosis of intracranial disease but occasionally they are encountered. In many foreign countries hydatid disease must always be considered. It would be wise for us also to remember this possibility whenever we have patients of foreign birth. All of the cases of intracranial echinococcus infection which I have seen have been born abroad, usually in Greece."

(Incidentally I remember helping Doctor Bailey at the operating table one day, with a patient who was thought to have a tumor of the cerebellum. As the cerebellum was exposed and the tonsils were spread, there suddenly appeared in the fourth ventricle a little cyst-like structure which he took hold of and said, "Ah, echinococcus!" and drew out of the fourth ventricle an echinococcus cyst.)

"Hydatid disease of man is caused by the cystic stage, in the intermediate host, of the taenia echinococcus which normally infests the intestine of dogs." (He doesn't say anything about donkeys.) "Man is infected by ingesting the ova; the embryo is hatched in the stomach or upper intestine and penetrates its wall. It is, therefore, understandable that the cysts are more frequent in the liver than in any other organ, since it is the first organ to which the venous blood carries the embryos. In man the cysts of the brain may be either primary or secondary. In the former case the embryo passes through the liver and lungs and lodges in the brain. Such primary cysts of the brain are rare and much more frequent in children than in adults. They are almost always solitary and may reach an enormous size, containing as much as nineteen ounces of fluid. The cyst has a very thin wall and causes very little reaction in the surrounding cerebral tissue. It is almost always in the cerebral hemisphere. The secondary cysts are metastatic and usually multiple. They may occur anywhere in the brain. They result from the transportation of scolices or brood-capsules to the brain following rupture of a primary cyst usually in the left side of the heart. The cysts are typically in the cerebral substance but may rupture secondarily into the subarachnoidal space or ventricles.

It can be understood that the symptoms resulting from such a disease of the brain will be quite varied. Almost any sort of focal symptom may occur, of which focal epilepsy is most common. Some of the patients are considered to be idiopathic epileptics, others as basilar luetic meningitis, some have no symptoms, the cysts being found at necropsy, and still others are psychotic—dementia, depression, disorientation, hallucinations, catatonia, manic excitement, and delirium, being observed. In children the cysts cause a peculiar erosion of the skull in their neighborhoods which is rather characteristic.

The diagnosis cannot be made from the neurological symptoms but is established by demonstrating a general hydatid disease. The cysts may be demonstrated in the liver or lungs. If not, the presence of such an infection may be established by complement-fixation test on the blood (positive in about 80 per cent of cases) or by intradermal test (positive in about 90 per cent to 95 per cent). No constant changes are produced in the cerebrospinal fluid although a paretic gold-curve may be found.

Precipitin and complement-fixation tests on this fluid are very inconstant and the Wassermann reaction is negative. The nationality and birthplace of the patient are of considerable importance. The disease is contracted usually in childhood and is especially prevalent in sheepherding communities where the hygienic conditions are not so good. It is most common in certain parts of Australia, South America, South Africa, and southeastern Europe."

Although we have an endemic focus in North Dakota, so far as I know there is not one in Texas, but all things considered, I would think that echinococcus infestation is the most likely diagnosis, either that or some other form of parasitic disease, and it would seem to me that there might be evidence of liver disease as was suggested by the findings, possibly pulmonary infestation as well. Other visceral changes I should expect to be secondary to his general condition of debility.

Dr. Sol Sherry: There were 30 students reporting. Insofar as the central nervous system lesion is concerned, 28 believed it was multiple sclerosis, one multiple cerebral thrombi secondary to lues, and one metastatic neoplasm probably secondary to the lung. Cysticercosis was considered by some but was deemed too rare. As far as the respiratory system was concerned, all the students believed that the patient had a pulmonary infarct. Twenty-five believed that there was suppurative pneumonia, and 5 a bronchial pneumonia without obvious suppuration. All believed that there was a urinary tract lesion. Twenty-six believed that there was probably a terminal septicemia.

Dr. Helen Glueck: Were there any skull films taken on this patient?

Dr. Levin: No.

Dr. Ferris: This difference of student opinion ought to raise some comment.

Student: Would Dr. Evans speculate on the possibility of gummatous lesions.

Dr. Evans: I would eliminate these for the same reason that I think the diagnosis of lues in general is unlikely.

Student: Why would you eliminate lues in general?

Dr. Evans: In the first place it doesn't appear to me to be the usual picture one sees with paresis or tabes or tabo-paresis. Secondly, there was obviously increased intracranial pressure at the time of the first hospital admission. It's a pity that he wasn't in condition for a neurological work-up on the second admission, but I would think that that was a valid increase in intracranial pressure as indicated by the two taps. Furthermore, the statistical odds where the blood and spinal fluid are negative as they were, makes it increasingly unlikely. The negative blood and spinal fluid serology doesn't rule it out completely, but would make it rather unlikely, and I think the further course of the disease is really not typical of any of the luetic processes. I don't know about the multiplicity of gummata in central nervous system lues; we see so little of it now. In my own experience gummas are usually single. I don't know whether that's generally true or not.

Dr. Charles D. Aring: Within the central nervous system we've never seen them except singly, and I recall only one or two cases that have come to our laboratory since 1936.

Dr. Eugene B. Ferris: Dr. Evans, would you comment on the differential diagnosis of multiple sclerosis here? A good many of the students made that diagnosis.

Dr. Evans: It disturbed me when I heard Dr. Sherry's report on the student conference because I hadn't considered multiple sclerosis seriously. Again, influenced by the cerebrospinal fluid findings, it is true that there was a first-zone gold curve and there were diffuse lesions, but in view of the information which we have on the first hospital admission I would hardly think that one would have to consider multiple sclerosis too seriously.

Dr. Ferris: Do you ever see that many white cells in the cerebrospinal fluid in multiple sclerosis?

Dr. Evans: I would say it is unusual.

Dr. Aring: One sees this number of white cells occasionally.

Dr. J. Park Biehl: Dr. Evans, were you able to localize this lesion?

Dr. Evans: No, I said it was diffuse. We have a history of weakness of the left side at the time of the first admission. We are told of increased deep reflexes generally at that time, but more so on the left than on the right. The Babinski responses were bilateral, so I would think that even on that first admission the evidence of pyramidal tract involvement would suggest bilaterality. The mental confusion certainly isn't helpful from the standpoint of localization. Then there was evidence of cerebellar disturbance. Now whether or not the incontinence which he developed was a manifestation of spinal cord disturbance or whether it was related to his confusion and general disintegration, I can't say. There were focal seizures witnessed on the last hospital admission which had their origin in the hemisphere opposite to that involved ten years earlier. I think the process was quite diffuse, and that's one of the reasons why I entertained the diagnosis of sarcomatosis or carcinomatosis of the leptomeninges. There might very well be diffuse leptomeningeal involvement and possibly multiple parasitic foci.

Dr. Ferris: Is there any indication in the record why the diagnosis of pulmonary infarction was made? I'm not quite clear about the association of infarction with as much atelectasis as he had.

Dr. James Ward: Since it occurred in October I don't remember the case clearly. At that time he had gone into sudden shock with cyanosis. He had pain in his chest and had rales of the left lower lobe. The evidence would seem that he had had a

pulmonary infarct although the source wasn't obvious.

Dr. Ferris: Isn't it unusual to get this much atelectasis with infarction?

Dr. Ward: At that time the x-ray was read as pulmonary infarct.

Dr. Levin: I think it more likely pneumonia with atelectasis.

Discussion of Pathology

Dr. Paul Wozencraft: The findings in the viscera were about what one would expect. The patient was emaciated and there were large decubiti. Pulmonary edema was extensive and there was a small infarct in the left lower lobe about the size of the lesion shown in the x-ray. There was a mild acute cystitis but the kidneys were normal. Apparently the urinary infection was well controlled by antibiotic therapy.

The autopsy was done 8 hours after death. The brain weighed 1325 grams. The cerebral hemispheres were symmetrical. Both temporal poles and a portion of the gyrus rectus on the left were soft. Over the optic chiasm and extending backward over the brain stem there was a thick, whitish membrane which was so thick that it was difficult to isolate the chiasm, and the infundibulum and basilar artery were completely obscured by it. This extensive, severe leptomeningitis was found only at the base. Section of the larger arteries of the circle of Willis revealed them to be patent and only mildly arteriosclerotic.

Coronal sections at 1 cm. of the cerebral hemispheres and brain stem revealed marked dilatation of the ventricular system. Sections from the central nervous system examined microscopically showed extensive chronic leptomeningitis with scarring and calcification. There were numerous structures within the exudate which at first glance resembled degenerated veins. However, closer inspection revealed a peculiar serrated border. This feature is characteristic of the bladder wall of Cysticercus. No viable larvae were to be found. You may ask, can we prove that this reaction is not due to syphilis? I think we can. In the first place the most severe reaction was around the bladder remnants. Secondly, the inflammation extended just to the adventia of the vessels and did not involve them in any area.

The disease suffered by this man is not really rare, but only rare in our experience. There are approximately 3 million people in the world infested by Taenia solium. The brain lesion of Cysticercosis is mostly in the leptomeninges where there resulted the white, opaque thickening of the leptomeninges at the base of the brain, occluding the fourth ventricle and extending around the optic chiasm. The floor of the third ventricle bulged. There was a severe hydrocephalus.

We might review briefly the life cycle of Taenia solium. The worm normally lives in the intestine of man and a single worm may persist for 25 years or more. The eggs are viable in the soil for weeks. When ingested by hogs, they hatch within 24 to 72 hours and the larvae migrate throughout the body and form Cysticerci within 60 to 70 days which may remain viable in the hog muscle for many years. In man, if the eggs enter the stomach either by reversal peristalsis from the small intestine or by ingestion, the shell is digested, and the cycle seen in the hog takes place in man. The larvae migrate and form cysts.

Considered in terms of world distribution, this disease is not rare. The British[2] have found that it must be seriously considered in patients with seizures in adult life and who have lived in India. Now in this country there are many people who have spent time in India and other parts of Asia. Fortunately, measly pork is rare in the United States. I recently spoke with one of the inspectors in the Bureau of Animal Industry, and he reported that in his experience of 21 years he had seen only one instance of measly pork in St. Paul, Minnesota. In the first half of the 19th century 2 per cent of the autopsies performed in central Europe showed Cysticercosis somewhere in the body. In some parts of Mexico and South America[3] Cysticercosis of the brain is as common as tuberculous meningitis and measly pork is found in 4½ per cent of the hogs slaughtered. In Guadalajara 3 per cent of a large series of autopsies showed Cysticercosis, and 25 per cent of a series of brain tumors consisted of Cysticercus. Dr. Norman Stall[4] in 1947 published an article on the incidence of all kinds of parasitic infection. It's a very salty article entitled, "This Wormy World,"

and he estimates from available reports the prevalence of various types of infestation of worms in mankind on a world-wide basis. That is the source of my estimate of 3 million cases of Taenia solium infestation. Incidentally it is stated in that article that the Chinese produce 18,000 tons of ascaris eggs annually.

Clinical Diagnosis

Meningovascular syphilis.
Second and third degree burns.
Atelectasis, left lower lobe.
Malnutrition.
Extensive decubital ulceration.

Dr. Evans' Diagnosis

Parasitic disease of the brain, probably echinococcus.
Pulmonary infarction with atelectasis.
Paralytic ileus.

Anatomical Diagnosis

Cysticercosis of the central nervous system.
Pulmonary infarct left lower lobe.
Extensive decubital ulceration.

Dr. Edward Gall: Dr. Evans, would you like to defend your failure to mention Cysticercosis in this case?

Dr. Evans: I cannot defend it, but I do have an explanation and a confession. I usually have some idea of what is going on in the laboratory of neuropathology. One day a member of the resident staff in pathology met Drs. Wozencraft and Shaver outside the door of the neuropathology laboratory and said in great excitement, "Do you know what we found in that case?" I was thinking about something else and half heard what he said. On first going over this protocol I realized that this was not likely to be lues of the central nervous system and that it might be a parasitic disease. Then it occurred to me that this might be the same case that the resident had talked about, but as I got working it over, I couldn't remember whether he had said Taenia solium or echinococcus. I have seen two cases of echinococcus involvement of the central nervous system, and I have seen only one of Cysticercosis, so echinococcus disease finally won out. Being reminded of that one case made me recall seeing on Gordon Holmes' service at the National Hospital, London, a case of Cysticercosis from India. In that particular case it was possible to demonstrate calcifications in the deltoid muscle and also, I think, in the muscles of the lower leg. I wonder if we would have any information from the X-ray Department to help us on that score?

Dr. Levin: We have no other films besides those demonstrated.

Dr. Aring: I think Dr. Evans' last point ought to be kept in mind. One of the best diagnostic tools in Cysticercosis is the x-ray. As far as I know, roentgen study rarely fails to demonstrate these calcified lesions, usually in muscles. Of course, one must suspect the diagnosis first; as I recall, it was quite routine to require such x-rays at the National Hospital, London, in an adult developing convulsive seizures, who had been abroad, especially to India.

Dr. Wozencraft: I'd like to ask either Dr. Aring or Dr. Evans their experience with eosinophilia in the cerebrospinal fluid? Dr. Lee Foshay mentioned the other day that eosinophilia of the spinal fluid is a strong indication of parasitic infestation of the central nervous system.

Dr. Aring: I can't recall ever having seen eosinophiles in the cerebrospinal fluid, but we do not do the Wright stain.

Dr. Frank R. Dutra: I have seen such eosinophilia in trichinosis where there is central nervous system manifestation.

Dr. Ferris: Was there any involvement of the liver?

Dr. Wozencraft: No, we did not find Cysticerci in any of the other organs. When the autopsy was done we were not aware that the patient had Cysticercosis, and no search was made for them; one probably should take more muscle, and the subcutaneous tissue is said to be fertile ground for their growth too. Frequently the finding of subcutaneous nodules has helped in pointing toward the diagnosis.

Student: Did he have this worm in the intestinal tract?

Dr. Gall: No.

Dr. Stanley E. Dorst: Is it your idea, Dr. Wozencraft, that this represents burned out disease, no longer active?

Dr. Wozencraft: In the sections that we have, the worms are not alive, but the reaction of the tissues usually does not begin until the larva is dead. There is active inflammation in the meninges.

Dr. Ferris: Would it have been possible to have missed the worm in the intestinal tract?

Dr. Wozencraft: I would doubt it. The adult worm attains a length of 2 to 7 meters. This patinet has harbored Cysticerci in his brain for more than ten years without necessarily having reinfestation.

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Transactions of the Galveston Neurological Society
March 28, 1952

IRA JACKSON, M.D., *Presiding*

PANCREATIC ADENOMA AS A CAUSE OF RECURRENT CEREBRAL DYSFUNCTION

ISRAEL H. SCHULEMAN, M.D.*

Symptoms due to a pancreatic adenoma often have been erroneously diagnosed as brain tumor, idiopathic epilepsy, acute alcoholism, encephalitis, neurocirculatory asthenia and psychosis. Harris in 1924 was the first to emphasize the fact that a fairly characteristic group of symptoms experienced by some patients is the result of a spontaneous fall of the blood sugar to hypoglycemic levels. Of the multiple causes of spontaneous hypoglycemia, three of the entire group are responsible for 80 per cent of the cases. They consist of: (1) functional hyperinsulinism; (2) organic hyperinsulinism; (3) hepatogenic hypoglycemia. The different types of hypoglycemia can be determined by (a) the level of the fasting blood sugar on normal carbohydrate feeding and on restriction of dietary carbohydrate or after a short fasting period; (b) the dextrose tolerance test after standard dietary preparation; (c) the result of several liver tests; (d) the history and clinical course of the disturbance.

Case Presentations

Case I: This patient was a 55 year old female with a history of spells for five years. The attacks may begin with sweating and generalized numbness followed by diminished hearing, vision and a sensation as though she were in a daze. The general physical and neurological examination were essentially normal except for obesity. The EEG, blood and urine studies were within normal limits. The six hour glucose tolerance test revealed 55 mgm. per cent blood sugar at four hours, accompanied by euphoria and confusion. On a starvation regime patient had a spell after thirty-six hours and the blood sugar level was 55 mgm. per cent. At operation, an adenoma of the pancreas was found and removed by Dr. A. O. Singleton, Jr. Patient made a good recovery and when last seen was free from spells.

Case II: This 51 year old female had a history of spells for four years beginning with visual blurring, numbness of the tongue, headache and vomiting. The attacks could be aborted by eating. If allowed to go on, she became confused and sleepy. The general physical and neurological examination was essentially normal. EEG and routine laboratory studies were within normal limits. The six hour glucose tolerance test revealed a level of 35 mgm. per cent blood sugar at five hours and was accompanied by a typical spell. On a starvation regime the patient had a spell after sixteen hours and the blood sugar level was 49 mgm. per cent. At operation an adenoma of the tail of the pancreas was found and removed by Dr. Singleton. The pathologist reported the tissue as having features suggesting the potentiality of being malignant. The patient when last seen was free from spells.

Case III: This 58 year old female presented a three year history of spells. The attacks were described as beginning with diplopia, followed by drowsiness. The general physical and neurological examination was essentially normal. The routine laboratory studies were within normal limits. The EEG was normal. The six hour glucose tolerance test revealed a level of 46 mgm. % of blood sugar but without noticeable symptoms. Patient fasted for fifty-six hours with no reaction. However, her blood sugar was found to be 37 mgm. % during a spontaneous spell. At operation a pancreatic adenoma was found and removed. When last seen the patient was free from spells.

Discussion

Dr. A. O. Singleton, Jr.: If the diagnosis of islet cell tumor of the pancreas cannot be ruled out, the patient should be explored. The head of the pancreas should be mobi-

*Asst. Prof. Neuropsychiatry University of Texas, Galveston.

lized by freeing up the lateral border of the second part of the duodenum and then carefully examined. The exploration of the posterior surface of the body and tail of the pancreas may be facilitated by removing the spleen and reflecting the body and tail of the pancreas with the splenic vessels at its superior border en mass to the right. Many small fragile vessels enter the pancreas from the splenic artery and vein, and if one tries to separate the pancreas from these while reflecting it, much troublesome bleeding results.

The finding of one adenoma should not deter the operator from making a complete examination because 10-20 per cent of lesions are multiple. Where no tumor can be found ectopic pancreatic tissue should be searched for, as 1-2 per cent of tumors are found outside of the pancreas.

There is one group of tumors which has caused concern. These appear benign grossly, and clinically do not appear to recur either locally or as distal metastases, but histologically appear malignant. These as well as the benign adenoma group usually have been treated by local excision with good results. True islet cell carcinoma has a very bad prognosis.

Where no tumor has been found many surgeons have blindly resected portions of the pancreas usually the body and tail as 70 per cent of adenomas are found in this region. This has resulted in improvement in about 50 per cent of cases, islet cell adenomas being found deep in the resected specimens in some instances. After removal of the tumor a diabetic state exists for a few days as the secretion of the normal pancreatic tissue has been supressed by the lesion.

At John Sealy Hospital, six cases have been explored for organic hyperinsulinism with the finding of adenomas in five cases. The adenomas were removed with relief of symptoms in all cases. The remaining case revealed no pancreatic pathology at exploration, but at autopsy the pituitary gland was found to have been replaced by metastatic carcinoma from an old breast cancer.

Dr. Hamilton Ford: The first case presented here is of interest because exploration may well have not been done. Although, she gave a classical history, at first no low fasting blood sugars could be obtained and even a 48 hour fast brought out no definite symptoms. These cases must be followed very closely and repeatedly checked or some of the more atypical ones may escape us. Often the relief of emotional tension and lack of physical activity which may occur when the patient is hospitalized may prevent attacks of hyperinsulinism.

Book Reviews

NEUROSURGERY: AN HISTORICAL SKETCH by *Gilbert Horrax*. Published by Charles C. Thomas. 142 pages.

Although neurosurgery is considered one of the newest specialties in medicine, Dr. Horrax has traced its origin to the Stone Age. Trepanation of the skull has been shown to be the oldest major surgical procedure. In the prehistoric and Egyptian era trepanation was performed for trauma, epilepsy, syphilis, melancholia and other disorders. The author briefly discusses the neurosurgical aspects of medicine in various eras to 1890. The year 1886 is noteworthy because of the founding of the Neurological Society of London with Hughlings Jackson as its first president, and the appointment of Sir Victor Horsley as surgeon at the National Hospital at Queen's Square. It was there that neurological surgery was born as a specialty in medicine with Horsley as it father. His last chapter, comprising over half of the book, covers the period from 1890 to the present.

The adjuncts both in technique and in diagnosis in neurosurgery are discussed. No such book would be complete without mention of the contributions of such men as Cushing, Dandy, de Martel, Vincent, Fraser, and Sacks. Surely no other two men have done more to bring the mortality rate of neurosurgery down to a respectable level than Dandy and Cushing. One might take exception to Horrax's concluding statement that operative technique must remain a *sine qua non*, for it has been the neurophysiological surgeon who has brought about the tremendous advances in the last fifty years.

This monograph is a worthy addition to the well known "American Lecture Series in Surgery." For such a concise and entertainingly written book it is admirably documented with many illustrations and an extensive bibliography.

BRAIN TUMORS OF CHILDHOOD by *Henry M. Cuneo, M.D.*, and *Carl W. Rand, M.D.* Charles C. Thomas, Publisher. 222 pages.

This concise well documented book is one of the excellent monographs of the "American Lecture Series in Surgery." The authors reviewed their own 83 patients with intracranial neoplasms and granulomatous lesions covering a ten year period. The subject matter is presented according to pathological entities. It might have been more appealing had the divisions been according to cerebral localization. Each type of tumor is dealt with from a histological, clinical, and therapeutic point of view. The chapters are introduced by a brief but thorough review of the literature. They do not burden the reader with many lengthy case reports except in unusual lesions where they are most needed. The two chapters on granulomatous lesions are most welcome and add to the value of this monograph. The bibliography is adequate. The photomigrographs and photographs of the gross pathology are well done as are the roentgen reproductions. Charles C. Thomas again has done a superb job of publishing.

Dr. Ira J. Jackson,
University of Texas Medical Branch, Galveston, Texas.

RESEARCH MEETING

The Annual Meeting of the Association for Research in Nervous and Mental Disease will be held on December 12 and 13, 1952 at the Hotel Roosevelt, New York City. The subject of the meeting will be "Metabolic and Toxic Diseases of the Nervous System."

VA COURSE IN PSYCHIATRY AND NEUROLOGY

The Veterans Administration is instituting a four-month intensive training course in psychiatry and neurology to fit the needs of physicians without such previous training who are assigned to duty in 22 predominantly psychitric hospitals. Physicians who have been engaged in general practice may request this training upon applying for a position at one of these hospitals.

The course will be held at the VA Hospitals in Coatesville, Pennsylvania; Palo Alto, California; and a joint Downey-Hines, Illinois, program near Chicago, Illinois. Physicians will be employed at salaries commensurate with their training and experience (salary range: $5,500 to $11,800 per annum) and assigned to the course with travel and per diem for the four-month period.

Information and applications may be obtained from your nearest VA Hospital or Regional Office, or by writing to the Chief Medical Director, Veterans Administration Central Office, Washington 25, D.C.

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